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 NEWS  1          Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Apr 08  "Ask CAS" for self-help around the clock
 NEWS  3  Apr 09  BEILSTEIN: Reload and Implementation of a New Subject Area
 NEWS  4  Apr 09  ZDB will be removed from STN
 NEWS  5  Apr 19  US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
 NEWS  6  Apr 22  Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
 NEWS  7  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS  8  Apr 22  Federal Research in Progress (FEDRIP) now available
 NEWS  9  Jun 03  New e-mail delivery for search results now available
 NEWS 10  Jun 10  MEDLINE Reload
 NEWS 11  Jun 10  PCTFULL has been reloaded
 NEWS 12  Jul 02  FOREGE no longer contains STANDARDS file segment
 NEWS 13  Jul 22  USAN to be reloaded July 28, 2002;
                  saved answer sets no longer valid
 NEWS 14  Jul 29  Enhanced polymer searching in REGISTRY
 NEWS 15  Jul 30  NETFIRST to be removed from STN
 NEWS 16  Aug 08  CANCERLIT reload
 NEWS 17  Aug 08  PHARMAMarketLetter(PHARMAML) - new on STN
 NEWS 18  Aug 08  NTIS has been reloaded and enhanced
 NEWS 19  Aug 19  Aquatic Toxicity Information Retrieval (AQUIRE)
                  now available on STN
 NEWS 20  Aug 19  IFIPAT, IFICDB, and IFIUDB have been reloaded
 NEWS 21  Aug 19  The MEDLINE file segment of TOXCENTER has been reloaded
 NEWS 22  Aug 26  Sequence searching in REGISTRY enhanced
 NEWS 23  Sep 03  JAPIO has been reloaded and enhanced
 NEWS 24  Sep 16  Experimental properties added to the REGISTRY file
 NEWS 25  Sep 16  Indexing added to some pre-1967 records in CA/CAPLUS
 NEWS 26  Sep 16  CA Section Thesaurus available in CAPLUS and CA
 NEWS 27  Oct 01  CASREACT Enriched with Reactions from 1907 to 1985
 NEWS 28  Oct 21  EVENTLINE has been reloaded
 NEWS 29  Oct 24  BEILSTEIN adds new search fields
 NEWS 30  Oct 24  Nutraceuticals International (NUTRACEUT) now available on STN
 NEWS 31  Oct 25  MEDLINE SDI run of October 8, 2002

 NEWS EXPRESS  October 14 CURRENT WINDOWS VERSION IS V6.01,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 01 OCTOBER 2002
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=> file .gary
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FULL ESTIMATED COST 0.21 0.21

FILE 'MEDLINE' ENTERED AT 11:41:31 ON 08 NOV 2002

FILE 'CANCERLIT' ENTERED AT 11:41:31 ON 08 NOV 2002

FILE 'BIOSIS' ENTERED AT 11:41:31 ON 08 NOV 2002
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L1 7575 GLI OR GLI1

=> s l1 and glioma
L2 124 L1 AND GLIOMA

=> s l2 and (antagon? or inhib? or prevent?)
L3 11 L2 AND (ANTAGON? OR INHIB? OR PREVENT?)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 6 DUP REM L3 (5 DUPLICATES REMOVED)

=> d ibib abs 1-6

L4 ANSWER 1 OF 6 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002419251 MEDLINE
DOCUMENT NUMBER: 22155304 PubMed ID: 12165511
TITLE: Sonic hedgehog promotes cell cycle progression in activated peripheral CD4(+) T lymphocytes.
AUTHOR: Lowrey Jacqueline A; Stewart Gareth A; Lindey Susannah; Hoyne Gerard F; Dallman Margaret J; Howie Sarah E M; Lamb Jonathan R
CORPORATE SOURCE: Immunobiology Group, Medical Research Council Center for Inflammation Research, University of Edinburgh Medical School, Edinburgh, United Kingdom.. J.A.Lowrey@ed.ac.uk
SOURCE: JOURNAL OF IMMUNOLOGY, (2002 Aug 15) 169 (4) 1869-75. Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020814
Last Updated on STN: 20020904
Entered Medline: 20020903
AB Sonic hedgehog (Shh) signaling is important in the growth and differentiation of many cell types and recently has been reported to play

a role in T cell development in the thymus. This prompted us to investigate whether or not Shh contributes to the clonal expansion of peripheral CD4(+) T cells. In this study, we demonstrate that Shh and other components of the signaling pathway patched, smoothened, and **Gli1** (**glioma**-associated oncogene) are expressed in peripheral CD4(+) T cells. The addition of the biologically active amino-terminal Shh peptide had no effect on resting CD4(+) T cells, but significantly enhanced proliferation of anti-CD3/28 Ab-activated CD4(+) T cells. This was not due to antiapoptotic effects, but by promoting entry of T cells into the S-G(2) proliferative phase of the cell cycle. Neutralizing anti-Shh Ab reduced T cell proliferation by **inhibiting** cell transition into the S-G(2) phase, suggesting that endogenously produced Shh plays a physiological role in the clonal expansion of T cells. Furthermore, we have observed a significant up-regulation of Shh and **Gli1** (**glioma**-associated oncogene) mRNA in activated CD4(+) T cells with or without addition of exogenous Shh, which corresponds with maximal CD4(+) T cell proliferation, whereas bcl-2 was only up-regulated in activated cells in the presence of Shh. Our findings suggest that endogenously produced Shh may play a role in sustaining normal CD4(+) T cell proliferation and exogenously added Shh enhances this response.

L4 ANSWER 2 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1999:17907 SCISEARCH
THE GENUINE ARTICLE: 148YL
TITLE: CDK4 gene amplification in osteosarcoma: Reciprocal relationship with INK4A gene alterations and mapping of 12q13 amplicons
AUTHOR: Wei G; Lonardo F; Ueda T; Kim T; Huvos A G; Healey J H; Ladanyi M (Reprint)
CORPORATE SOURCE: MEM SLOAN KETTERING CANC CTR, DEPT PATHOL, 1275 YORK AVE, NEW YORK, NY 10021 (Reprint); MEM SLOAN KETTERING CANC CTR, DEPT PATHOL, NEW YORK, NY 10021; MEM SLOAN KETTERING CANC CTR, DEPT HUMAN GENET, NEW YORK, NY 10021
COUNTRY OF AUTHOR: USA
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (18 JAN 1999) Vol. 80, No. 2, pp. 199-204.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0020-7136.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 40

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The INK4A gene, localized to human chromosome 9p21, encodes p16(INK4A), a tumor suppressor that functions at least in part through the **inhibition** of CDK4, a cyclin-dependent kinase encoded by a gene at 12q13. To examine INK4A gene alterations in uncultured samples of osteosarcoma and the relationship between INK4A and CDK4 alterations, we analyzed the INK4A and CDK4 genes in 87 specimens from 79 patients. INK4A deletion and CDK4 gene amplification were determined by quantitative Southern blot analysis. INK4A exon 2 was screened for mutation by polymerase chain reaction and single-strand conformational polymorphism analysis. Methylation at the CpG island in INK4A, associated with loss of p16(INK4A) expression, was assessed by Southern blot analysis using methylation-sensitive restriction enzymes. INK4A deletion (4/55) or rearrangement (1/55) was found in 5 of 55 cases. No INK4A exon 2 point mutations and methylation were detected. CDK4 gene amplification was found in 6 of 67 samples, but not in tumors with INK4A alteration. Amplification analysis of other genes at 12q13 (**GLI**, CHOP, HMGI-C and MDM2) in these 6 cases supports the view that CDK4 and MDM2 are independent targets for amplification, with variable amplification of the intervening region containing HMGI-C. Of 46 patients studied for both INK4A alterations and CDK4 amplification, the tumors in 22% contained one or the other. The

prevalence of these alterations, in conjunction with the reported inactivation of RE in up to 80% of cases, suggests that genetic lesions deregulating the G(I) to S cell cycle checkpoint may be an almost constant feature in the pathogenesis of osteosarcoma. (C) 1999 Wiley-Liss, Inc.

L4 ANSWER 3 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:867103 SCISEARCH
THE GENUINE ARTICLE: 137GK
TITLE: Preliminary characterization of glial-secreted factors responsible for the induction of high electrical resistances across endothelial monolayers in a blood-brain barrier model
AUTHOR: Ramsohoye P V (Reprint); Fritz I B
CORPORATE SOURCE: BABRAHAM INST, DEPT CELL PHYSIOL, CAMBRIDGE CB2 4AT, ENGLAND (Reprint)
COUNTRY OF AUTHOR: ENGLAND
SOURCE: NEUROCHEMICAL RESEARCH, (DEC 1998) Vol. 23, No. 12, pp. 1545-1551.
Publisher: PLENUM PUBL CORP, 233 SPRING ST, NEW YORK, NY 10013.
ISSN: 0364-3190.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 21
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Factors secreted by C6 **glioma** cells which induce electrical resistances across endothelial monolayers in an in vitro blood-brain barrier model have been partially characterised for the first time. These transendothelial electrical resistances (TEERs) were only evident when cell-free conditioned medium derived from C6 **gli** oma cells was applied to the basolateral surfaces of confluent ECV304 or ECV304-9 cells which are both human umbilical vein endothelial cell lines (HUVEC). Electrical resistance values as high as 600 ohm. sq cm were obtained with this blood-brain barrier model and ultrafiltration techniques suggest that any factor(s) in the conditioned medium responsible for these TEERs have molecular masses of less than 1000 Da. Enzymic proteolysis and heat treatment carried out on the conditioned medium failed to **inhibit** its effect on the HUVEC monolayers suggesting that these CG cell-secreted factors are unlikely to be proteins.

L4 ANSWER 4 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
ACCESSION NUMBER: 1998:49967 BIOSIS
DOCUMENT NUMBER: PREV199800049967
TITLE: Molecular changes during the genesis of human **gliomas.**
AUTHOR(S): Sehgal, Anil (1)
CORPORATE SOURCE: (1) Pacific Northwest Cancer Found., 120 Northgate Plaza, Room 230, Seattle, WA 98125 USA
SOURCE: Seminars in Surgical Oncology, (Jan.-Feb., 1998) Vol. 14, No. 1, pp. 3-12.
ISSN: 8756-0437.
DOCUMENT TYPE: General Review
LANGUAGE: English

AB Neoplastic transformation in the normal human brain occurs as a result of the accumulation of a series of genetic alterations. These genetic alterations include the loss, gain or amplification of different chromosomes which lead to altered expression of proteins that play important roles in the regulation of cell proliferation. Several common genetic alterations at the chromosomal level (loss of 17p, 13q, 9p, 19, 10, 22q, 18q and amplification of 7 and 12q) have been observed. These alterations lead to changes in the expression of several genes; protein 53 (p53), retinoblastoma (RB), interferon (INF)alpha/beta, cyclic AMP dependent kinase number 2 (CDKN2), mutated in multiple advanced cancers 1 (MMAC1), deleted-in-colon carcinoma (DCC), epidermal growth factor

receptor (EGFR), platelet derived growth factor (PDGF), platelet derived growth factor receptor (PDGFR), MDM2, **GLI**, CDK4 and SAS during the genesis and progression of human **gliomas.** Recent studies suggest that altered expression of several other genes (MET-, MYC; transforming growth factor beta (TGFbeta); CD44; vascular endothelial growth factor (VEGF); human neuroglial-related cell adhesion molecule (hNr-CAM); neuroglial cell adhesion molecule (NCAM LI); p21waf1/Cip1; TRKA; mismatch repair genes (MMR); C4-2; D2-2) and proteins (e.g., cathepsins, tenascin, matrix metalloproteases, tissue **inhibitors** of metalloproteases, nitric oxide synthase, integrins, interleukin-13 receptor (IL-13R) Connexin43, urokinase-type plasminogen activator receptors (uPARs), extracellular matrix proteins and heat shock proteins) are associated with the genesis of human **gliomas.** Taken together, these findings point to the accumulation of multiple genetic mutations coupled with extensive changes in gene expression in the etiology of human **gliomas.**

L4 ANSWER 5 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97372837 EMBASE
DOCUMENT NUMBER: 1997372837
TITLE: Molecular changes during the genesis of human **gliomas.**
AUTHOR: Sehgal A.
CORPORATE SOURCE: Dr. A. Sehgal, Pacific Northwest Cancer Foundation, 120 Northgate Plaza, Seattle, WA 98125-7001, United States. asehgal@nwhsea.org
SOURCE: Seminars in Surgical Oncology, (1997) 14/1 (3-12). Refs: 83
ISSN: 8756-0437 CODEN: SSONEV
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
016 Cancer
022 Human Genetics
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Neoplastic transformation in the normal human brain occurs as a result of the accumulation of a series of genetic alterations. These genetic alterations include the loss, gain or amplification of different chromosomes which lead to altered expression of proteins that play important roles in the regulation of cell proliferation. Several common genetic alterations at the chromosomal level (loss of 17p, 13q, 9p, 19, 10, 22q, 18q and amplification of 7 and 12q) have been observed. These alterations lead to changes in the expression of several genes; protein 53 (p53), retinoblastoma (RB), interferon (INF)α/β, cyclic AMP dependent kinase number 2 (CDKN2), mutated in multiple advanced cancers 1 (MMAC1), deleted-in-colon carcinoma (DCC), epidermal growth factor receptor (EGFR), platelet derived growth factor (PDGF), platelet derived growth factor receptor (PDGFR), MDM2, **GLI**, CDK4 and SAS during the genesis and progression of human **gliomas.** Recent studies suggest that altered expression of several other genes [MET; MYC; transforming growth factor β (TGFβ); CD44; vascular endothelial growth factor (VEGF); human neuroglial-related cell adhesion molecule (hNr-CAM); neuroglial cell adhesion molecule (NCAM L1); p21(Waf1)/(Cip1); TRKA; mismatch repair genes (MMR); C4-2; D2-2] and proteins [e.g., cathepsins, tenascin, matrix metalloproteases, tissue **inhibitors** of metalloproteases, nitric oxide synthase; integrins, interleukin-13 receptor (IL-13R), Connexin43, urokinase- type plasminogen activator receptors (uPARs), extracellular matrix proteins and heat shock proteins] are associated with the genesis of human **gliomas.** Taken together, these findings point to the accumulation of multiple genetic mutations coupled with extensive changes in gene expression in the etiology of human **gliomas.**

L4 ANSWER 6 OF 6 CANCERLIT

ACCESSION NUMBER: 96603376 CANCERLIT
DOCUMENT NUMBER: 96603376
TITLE: Correlation of in vitro antitumor activity of irinotecan and topoisomerase I activity and levels in brain tumor (Meeting abstract).
AUTHOR: Savaraj N; Xu R; Wu C J; Landy H; Chua L; Solomon J; Feun L
CORPORATE SOURCE: V.A. Medical Center, Miami, FL 33125.
SOURCE: Proc Annu Meet Am Soc Clin Oncol, (1995) 14 A1610.
ISSN: 0732-183X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199604
ENTRY DATE: Entered STN: 19970509
Last Updated on STN: 19970509

AB Camptothecin (CPT), a topoisomerase I (TOP1) **inhibitor**, has been shown to have activity in brain tumor (BT). Recent pharmacokinetic data of CPT analogs suggest these compounds can penetrate the CNS quite readily. In this study, we investigated the in vitro antitumor activity of CPT analog, irinotecan (CPT-11), and its active metabolite SN-38 in 3 **glioma** cell lines. In addition, we attempted to study whether the TOP1 activity/levels can predict the sensitivity of these cells to CPT-11 or SN-38. Relaxation of supercoiled DNA was used to assay TOP1 activity and western blot analysis for TOP1 levels. The results are shown in a table. The addition of U-74500A (kindly provided by Upjohn), a 21 aminosteroid which has been shown to decrease brain edema, **inhibit** lipid peroxidation and reduce membrane ion transport enhanced the antitumor activity of SN-38 in all three cell lines. The IC50 of **gli**, U118, and U373 was reduced to 18, 9, and 8 nM respectively. Our data suggest that SN-38 has antitumor activity in BT and there is a potential correlation of TOP1 activity X levels with cytotoxicity of SN-38. Also, U-74500A can potentiate the antitumor activity of SN-38.

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 NEWS 20  Aug 19  IFIPAT, IFICDB, and IFIUDB have been reloaded
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               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 01 OCTOBER 2002
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       BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
       CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,
       DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...'
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64 FILES IN THE FILE LIST IN STNINDEX

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           2   FILE ADISALERTS
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          23   FILE BIOSIS
          14   FILE BIOTECHNO
           1   FILE CABA
          28   FILE CANCERLIT
          16   FILE CAPLUS
           4   FILE DDFU
           2   FILE DRUGNL
           6   FILE DRUGU
           2   FILE EMBAL
          15   FILE EMBASE
           5   FILE ESBIOBASE
          12   FILE FEDRIP
           1   FILE JICST-EPLUS
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          11   FILE LIFESCI
          25   FILE MEDLINE
          12   FILE PASCAL
           1   FILE PHAR
           3   FILE PHIN
           5   FILE PROMT
          25   FILE SCISEARCH
           1   FILE TOXCENTER
          33   FILE USPATFULL
           1   FILE USPAT2
           2   FILE WPIDS
           2   FILE WPINDEX

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L1   QUE GLI AND GLIOMA

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FULL ESTIMATED COST                                         1.06       1.27

FILE 'USPATFULL' ENTERED AT 12:00:16 ON 08 NOV 2002
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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 7 Nov 2002 (20021107/PD)
FILE LAST UPDATED: 7 Nov 2002 (20021107/ED)
HIGHEST GRANTED PATENT NUMBER: US6477708
HIGHEST APPLICATION PUBLICATION NUMBER: US2002166154
CA INDEXING IS CURRENT THROUGH 7 Nov 2002 (20021107/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 7 Nov 2002 (20021107/PD)
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USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Aug 2002
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>>>  classifications, or claims, that may potentially change from      <<<
>>>  the earliest to the latest publication.                           <<<

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            21 GLIS
           259 GLI
                 (GLI OR GLIS)
          2214 GLIOMA
          1130 GLIOMAS
             1 GLIOMATA
          2754 GLIOMA
                 (GLIOMA OR GLIOMAS OR GLIOMATA)
L2          33 GLI AND GLIOMA

=> s l2 and (inhib? or antagon?)
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         40386 ANTAGON?
L3          30 L2 AND (INHIB? OR ANTAGON?)

=> s l3 and py<=1997
       2267902 PY<=1997
L4           2 L3 AND PY<=1997

=> d ibib abs 1-2

L4   ANSWER 1 OF 2  USPATFULL
ACCESSION NUMBER:        95:27395  USPATFULL
TITLE:                   Structural alterations of the EGF receptor gene in
                         human gliomas
INVENTOR(S):             Vogelstein, Bert, Baltimore, MD, United States
                         Bigner, Darell, Chapel Hill, NC, United States
PATENT ASSIGNEE(S):      The Johns Hopkins University, Baltimore, MD, United
                         States (U.S. corporation)
                         Duke University, Durham, NC, United States (U.S.
                         corporation)

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 5401828               19950328            <--
APPLICATION INFO.:       US 1992-991286           19921215  (7)
RELATED APPLN. INFO.:    Division of Ser. No. US 1990-627869, filed on 17 Dec
                         1990, now patented, Pat. No. US 5212290 which is a
                         division of Ser. No. US 1990-531410, filed on 1 Jun
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1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-404226, filed on 8 Sep 1989, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Lacey, David L.
ASSISTANT EXAMINER: Feisee, Lila
LEGAL REPRESENTATIVE: Banner, Birch, McKie & Beckett
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 24 Drawing Figure(s); 20 Drawing Page(s)
LINE COUNT: 1897
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The epidermal growth factor receptor (EGFR) gene is amplified in 40% of malignant **gliomas** and the amplified genes are frequently rearranged. The genetic alterations associated with these rearrangements are characterized in five malignant **gliomas.** In one tumor, the rearrangement resulted in the deletion of most of the extracytoplasmic domain of the receptor, resulting in a hybrid mRNA between new sequences and the truncated EGFR. The predicted amino acid sequence of the protein from this tumor was remarkably similar to that described for several viral erb-B oncogenes. Four other tumors were noted to have internal deletions of the EGF receptor gene. These rearrangements brought about in-frame deletions affecting either of two cysteine-rich domains in the extracytoplasmic portion of the molecule. The clonal nature of these alterations, and the fact that identical alterations were seen in more than one tumor, suggests a role for these mutant receptor proteins in tumorigenesis. Furthermore, these studies document the existence of tumor specific cell molecules resulting from somatic mutation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 2 OF 2 USPATFULL
ACCESSION NUMBER: 93:40113 USPATFULL
TITLE: Antibodies specific for type II mutant EGTR
INVENTOR(S): Vogelstein, Bert, Baltimore, MD, United States
Bigner, Darell, Chapel Hill, NC, United States
PATENT ASSIGNEE(S): The Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)
Duke University, Durham, NC, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5212290		19930518	<--
APPLICATION INFO.:	US 1990-627869		19901217 (7)	

RELATED APPLN. INFO.: Division of Ser. No. US 1990-531410, filed on 1 Jun 1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-404226, filed on 8 Sep 1989, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
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NUMBER OF CLAIMS: 3
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 29 Drawing Figure(s); 25 Drawing Page(s)
LINE COUNT: 1845
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polyclonal and monoclonal antibodies which recognize mutant epidermal growth factor receptors (EGFR) have been produced. The EGFR gene is amplified in 40% of malignant **gliomas** and frequently the amplified genes are rearranged. Internal deletions in four **glioma** cell lines created an epitope which was not present in

normal EGFR. These mutant were characterized as expressing mutant type 1I EGFR and were found in a small percent of **gliomas.** Antibodies against these epitopes are useful for di

This invention was made with the support of the National Institutes of Health. The United States Government retains certain rights in the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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